Beyond Delaney

Preventing Exposure to Hazardous Pesticides

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Beyond Delaney:
Preventing Exposure to Hazardous Pesticides

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The Science and Environmental Health Policy Project (SEHPP) works to promote the use of sound scientific data in setting and evaluating environmental policies that effect public health. SEHPP is headed by David Rall, M.D., Ph.D., former director of the National Institutes of Health National Institute of Environmental Health Sciences, and founder of the National Toxicology Program of the Department of Health and Human Services.

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Introduction

In 1958, for the first time, the principle of cancer prevention was put into law: Congress adopted the Delaney Clause, an amendment to the Federal Food Drug and Cosmetic Act of 1954 that prohibited the addition to processed foods of any chemical shown to induce cancer in animals or humans. Today, as the Congress is poised to gut food safety laws, it is time to strengthen the Delaney Clause even further.

The Delaney Clause has been very valuable, but it does not go far enough. The amendment applies only to processed foods, not to raw fruits and vegetables. This leads to contradictory results: in processed foods, no amount of a carcinogenic pesticide is permitted; in raw produce, however, the same carcinogenic pesticide is allowed if the economic benefits of using the pesticide are judged to outweigh the risk to health.

In the four decades since the amendment was adopted, we have steadily improved techniques for evaluating the toxicity of chemicals and we've now considerably advanced our understanding of cancer. For instance, we've learned that some carcinogens are more potent than others. The Delaney Clause, however, treats all carcinogens the same; it does not allow regulators first to ban carcinogens of greater potency and second to direct resources to less worrisome pesticides.

Moreover, new developments in toxicology have shown that cancer is not the only health effect that should be of concern. Some pesticides poison the nervous system and affect development, while others disrupt the hormone system and the immune system. Like carcinogens, non-carcinogenic pesticides also have the potential to adversely affect health at chronic low doses. By focusing only on cancer-causing chemicals, the amendment is too narrow in scope.

In 1958 the Delaney Clause was a milestone, but today it needs to be broadened to reflect our present scientific understanding and to better protect public health.

It's time to move beyond Delaney and strengthen our commitment to prevention.

Prevention makes sense in the long-run. Preventing disease, as opposed to treating sick people, saves health care dollars and is more humane, as childhood immunization programs have shown. Reducing the public's exposure to toxic chemicals is a wise approach to preventing environmental illness. For instance, federal bans on lead in gasoline, interior house paint and food cans have lowered blood lead levels in children around the country and will prevent neurological impairment in generations to come.

We can apply this same principle of prevention to food safety by adopting a new strategy that keeps the most toxic pesticides out of food. This is achievable now without giving up safe and effective uses of pesticides. That's because a relatively small number of carcinogenic, neurotoxic and hormonally active pesticides contribute a large fraction of the total hazard that pesticides pose to the public. We already have many alternatives to these particular chemicals and can find many more. There's no need to accept the health risks associated with their use.

Revised food safety legislation should require that the Environmental Protection Agency (EPA) identify the most toxic pesticides and phase them out of use within a reasonably short period of time. Furthermore, pesticides that remain on the market should meet a strict safety standard designed explicitly to protect infants and young children.

A new policy of this kind that aims to reduce public exposure to highly toxic pesticides would have another important benefit: the spread of these chemicals throughout the environment would decrease. That should save taxpayers money now spent on expensive, and rarely successful, efforts to clean up degraded air, soil, and water. Clearly, the public should not have to subsidize the use of massive quantities of highly toxic pesticides on crops and then pay again to remove these same chemicals from contaminated drinking water and foods.

Ultimately, pesticides must be considered part of the larger problem of pollution. Tens of thousands of different toxic chemicals — trillions of pounds of them — are dispersed into the envi-

ronment each year. Little is known about the human health effects of chronic exposure to such a dizzying array of chemicals, although there's evidence that related compounds often act additively and some chemicals even enhance one another's toxicity.

Common sense tells us that we cannot continue dumping tons of toxic compounds into the environment and expect to escape harming human health. As we already see in polluted areas of Eastern Europe and the former Soviet Union, as well as in parts of the U.S., exposure to toxic chemicals in air, water, and food is associated with increased incidence of certain cancers, birth defects, and a variety of diseases. Future generations will likely receive much greater exposures, if present practices continue.

In spite of our scientific advances, we still don't have complete toxicity data for a large fraction of the chemicals we release into the environment and we may never catch up with the testing. Existing data gaps make it extremely difficult, if not impossible, to determine the risks of chronic exposure to complex mixtures of chemicals. While we know that children receive higher dietary exposures to pesticides than do adults, we have scant data on the specific developmental effects of pesticides on the young.

Given such uncertainties, we should redirect our efforts toward preventing exposure. We can begin by blending common sense with good science to build upon and improve the Delaney Clause.

We must create a new policy of prevention that reduces the number of highly hazardous pesticides allowed in food.

It's time now to bring food safety into the 21st century.

Since the Delaney Clause was adopted, the number of pesticides available has grown and use is rising.

The Delaney Clause amended the Federal Food Drug and Cosmetic Act in 1958 to protect the public from exposure to carcinogens; it prohibited the addition to processed foods of any chemical shown to induce cancer in animals or humans. The amendment came in response to the growing use of pesticides and food additives.

The first pesticides to be synthesized by chemists were developed just after World War II, and ever since then, the number of different pesticide products has ballooned. Today there are about 600 active ingredients in 23,000 pesticide formulations registered with the Environmental Protection Agency (EPA). These chemical pesticides primarily include herbicides, which kill weeds, insecticides, which kill insects, and fungicides, which kill fungal pests. The agency estimates that approximately 270 pesticides are directly applied to food crops.

The amount of pesticides used on crops in the U.S. has risen dramatically during the past few decades. Between 1966 and 1991, the pounds per acre of active pesticide ingredients that were applied to croplands more than doubled, from 1.2 pounds per acre to 2.7 pounds per acre (Public Voice 1993, p.2). In the last decade, fungicide use doubled and herbicide use increased 23.1 percent (Public Voice, p.3).

By volume, insecticide use has declined primarily because chemicals such as toxaphene, which is applied at 3 to 9 pounds per acre, have been replaced with more potent insecticides such as fenvalerate and permethrin, which are applied at 0.5 pounds per acre. The fraction of U.S. cropland treated with insecticides increased between 1969 and 1987 from 15.4 percent to 24.4 percent (Public Voice, p. 4).

As of 1991, approximately 817 million pounds of active pesticide ingredients were used on crops: nearly two-thirds, or 495 million pounds, were herbicides; about one-fifth, or 175 million pounds were insecticides; and approximately one-tenth, or 75 million pounds, were fungicides (Public Voice, pp 2-3).

The Delaney Clause and other pesticide regulations have not prevented multiple pesticide residues from occurring in food.

Millions of pounds of pesticides are applied to crops each year, and some of these chemicals persist in raw foods. According to data from 1987-1989, fruits and vegetables receive the heaviest applications of herbicides, insecticides, and fungicides (Public Voice, p. 5).

An analysis by the Environmental Working Group (EWG) found that multiple residues of pesticides commonly occur on fruits and vegetables (EWG 1994a, pp. 1-3). Their analysis of USDA test results for 12 ready-to-eat fruits and vegetables revealed that more than 50 percent of peach, apple, and celery samples contained residues of more than one pesticide; 10 percent of the servings had four or more pesticides on them.

Moreover, EWG found that washing and preparing these samples for consumption did not remove the residues. The United States Department of Agriculture (USDA) found eight pesticides on individual samples of apples, seven on peaches, and six on grapes that were washed and prepared for normal consumption (USDA 1994). Indeed, many pesticides used today cannot be removed because they are systemic; rather than occurring just on the surface of fruits and vegetables, they are incorporated into all tissues of the plant.

Many Americans ingest pesticides in their drinking water.

A portion of the millions of pounds of pesticides applied to crops each year wash off into waterways or seep deep into soil and end up in drinking water. A 1990 EPA survey found that 10.4 percent of community water system wells and 4.2 percent of rural domestic wells contained one or more pesticides. Pesticides are generally not removed by the standard water treatment technologies used by most water companies.

In the Midwest Corn Belt region and in

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Louisiana, where pesticide use and run-off is heavy, it has been estimated that 11.7 million people drink water contaminated with five major herbicides — atrazine, cyanazine, simazine, alachlor, and metolachlor (EWG and Physicians for Social Responsibility 1994b, p.1). From 1987 to 1994, 67 pesticides and metabolites were detected in midwestern sources of drinking water.

Other sites of significant contamination of drinking water include: California, where soil fumigants DBCP and EDT used to control nematodes seeped into groundwater; Florida, where EDT and the carbamate insecticide aldicarb tainted wells; Long Island, New York, where aldicarb entered groundwater (Gustafson 1993, pp. 7-27).

Since the Delaney Clause was adopted, we've learned pesticides cause more health effects than originally thought.

CANCER. Exposure to certain pesticides increases the risk of developing cancer. Agricultural workers, who contacted relatively high levels of pesticides, show elevated rates of leukemia, Hodgkin's disease, multiple myeloma, skin melanomas, and cancers of the lip, stomach, and prostate, and statistically non-significant increases in non-Hodgkins lymphoma and cancers of connective tissue and brain (Blair 1992). Several studies have found an increased incidence of malignant lymphoma and soft tissue sarcoma in people occupationally exposed to phenoxy herbicides, such as 2,4-D and 2,4,5-T (Pearce 1990).

Recently, a disturbing trend has been spotted: many of the same cancers that are prevalent in farm workers appear to be increasing in the general population (Davis, Devra et al. 1992). For example, during the past 40 years the incidence of non-Hodgkins lymphoma has risen 172 percent, multiple myeloma 183 percent, and brain cancer 74 percent (Miller 1993).

Could exposure to pesticides be the common factor? It cannot be ruled out, since these chemicals have become ubiquitous.

Contrary to what industry tells the public, low-dose exposures to carcinogens should be of concern, particularly in light of the large number of chemicals now in the environment and the potential of carcinogens to act additively. Researchers generally believe that even small doses of cancer-causing chemicals present real risks (Portier et al. 1994). A review by the National Toxicology Program found that 6 percent of 195 chemicals analyzed increased cancer risk at high doses only (Haseman and Lockhardt 1994).

It's biologically plausible that widespread low-dose exposures to chemicals in the environment, including pesticides, could be contributing to rising rates of certain cancers in the general population (Landrigan 1992, Rall 1994). Tens of thousands of chemicals of unknown and untested carcinogenic potential have been released into the environment since World War II (NRC 1984).

Indeed, a recent study found that Caucasian baby boomers born between 1948 and 1957 have a significantly greater risk of developing cancer when compared with earlier generations, and the increase cannot be completely accounted for by smoking, aging, or better diagnostic tools (Davis, Devra et al. 1994). Baby boomers are the first generation to grow up in the era of synthetic chemicals. During the post-World War II period, breast cancer incidence has been increasing by about one percent per year and some researchers hypothesize that exposure to pesticides in the environment, such as DDT, may be a contributing factor (Davis, Devra et al. 1993).

Environmental factors are likely to play a significant role in the development of childhood cancers. A review of 32 studies on parental occupation and childhood cancer concluded that occupational exposure of parents to chemicals (paints, petroleum products, hydrocarbon solvents, metals, and pesticides) raises the risk of cancer in their children (O'Leary 1991). Workers often bring home chemicals on their skin and clothing.

Although mortality has declined, the incidence of the two most common childhood cancers is in fact rising: brain/nervous system cancer

increased by 32.6 percent and acute lymphocytic leukemia by 27.4 percent between 1973 and 1990 (Ries 1993). Cancer is now the second leading cause of death in children under age 14 (accidents are first).

The cause for this increase in childhood cancer remains a mystery. However, exposure to low levels of pesticides early in life may well be a contributing factor. A recent study of Denver children under age 15 found a strong association between home use of pesticides in the yard and soft-tissue sarcoma, and between use of pest strips and leukemia (Leiss 1995). Another study found a link between home use of pesticides and childhood brain cancer (Davis, James et al. 1993).

NEUROTOXICITY. The widely used insecticides known as organophosphates and carbamates are neurotoxic to insects. These compounds are designed to block the action of cholinesterase, an essential enzyme that influences the transmission of nerve signals. Unfortunately, these pesticides have the same effect in humans, causing a disturbance in the functioning of the nervous system. Acute poisoning from this class of chemicals can cause diarrhea, muscle twitching, visual disturbances, hypertension, bradycardia, mood swings, respiratory distress, and death. Indeed, acute poisoning from organophosphate and carbamate pesticides afflicts hundreds of thousands of people around the world each year (WHO 1990).

Farmworkers, who are often exposed chronically to organophosphates and carbamates, are more likely than non-farmworkers to show depressed cholinesterase activity, although the significance of this effect is still debated (Cieselski 1994). A study that examined the subclinical effects of low-level, long-term exposure to an organophosphate called fenthion found that agricultural workers in India showed abnormal nerve conduction velocities (Misra 1988).

Such subtle changes in the nervous system may well have long-term health consequences, particularly for exposed young children, whose brains and nervous systems are still developing. According to a report by the National Academy of Sciences (NAS), "The emerging data suggest that neurotoxic and behavioral effects may result from low-level chronic exposure to some organophosphate and carbamate pesticides" (NRC 1993a, p.64).

Indeed, some children may already be showing signs of acute neurotoxicity from dietary pesticides. The NAS report states that "for some children exposures could be sufficiently high to produce symptoms of acute organophosphate poisoning" (NRC 1993a, p.7). In these children, the symptoms of acute toxicity — headache, nausea, and irritability — would be difficult to distinguish from other common illnesses.

HORMONAL EFFECTS. It has only recently been discovered that a wide variety of pesticides can mimic or interfere with sex hormones. DDT, kepone, dieldrin, toxaphene, and chlordane — all bioaccumulative organochlorine insecticides used heavily in the past and ubiquitous in the food chain — are now known to be "environmental estrogens." Endosulfan, one of the most commonly used pesticides in the U.S. today, was recently found to be estrogenic (Soto 1994).

These compounds can bind to the estrogen receptor and mimic the action of the natural hormone, although it appears that environmental estrogens are less potent than the body's own estradiol. Pesticides that are estrogenic spur cell proliferation in cell culture and in certain target tissues of lab animals, just as estradiol does.

Not all pesticides with hormone-like properties mimic estrogen. Some act through other mechanisms — for instance, metabolites of the fungicide vinclozalin appear to block testosterone from binding to its receptor (Kelce 1994). Other compounds may alter the synthesis and metabolism of natural hormones or alter hormone receptor levels in tissues. Widely used endocrine-disrupting pesticides include the herbicides atrazine, cyanazine, 2,4-D, and 2,4,5-T. Many pesticides have not yet been thoroughly tested for endocrine activity.

The long-term effects of human exposure to pesticides with endocrine activity are largely

unknown. However, reproductive dysfunctions have been documented in a wide variety of exposed wildlife species, and any perturbation of the normal levels and functions of sex hormones in humans could potentially have serious health consequences (Colborn 1993).

Some researchers propose that exposure to estrogenic chemicals, including pesticides, may be a causative factor in a rise in reproductive disorders in men (Sharpe and Skakkebaek 1993). Male pesticide workers exposed to the nematocide DBCP suffered decreased testosterone, low sperm counts, and long-term sterility (Whorton 1979). There's evidence that sperm counts and semen volume have been declining in men during the past 20 years in Paris (Auger 1995) and during the past 50 years in other industrialized countries (Carlsen 1992). Since 1950, the rate of testicular cancer, which afflicts primarily younger men, has increased 125 percent in the U.S. (Miller 1993); two- to four-fold increases in incidence have been reported in other industrialized countries (Osterlind 1986).

Female reproductive cancers might also be influenced by pesticide exposures. One medical hypothesis suggests that estrogenic chemicals have contributed to the steady increase in breast cancer incidence since the 1940s (Davis 1993). An epidemiological study found a significant association between higher blood levels of DDE (the metabolite of the insecticide DDT) and increased risk of breast cancer in Caucasian women (Wolff 1993). Another study linked high blood levels of DDE with elevated risk of breast cancer in Caucasian and African American women but not Asian women (Krieger 1994). The National Cancer Institute has begun a project to examine the relationship between exposure to pesticides and risk of breast cancer in suburban Long Island women.

Endocrine-disrupting pesticides pose a greater hazard to embryos and fetuses, whose development depends on a finely regulated balance of hormones. Sex hormones direct the embryonic and fetal development of the reproductive system and greatly influence the emerging immune and nervous systems. A disturbance in the normal hormone balance in the womb could lead to life-long deficits in any or all of these systems. A study of rats exposed pre- and post-natally to low doses of chlordane found abnormal sexual behaviors and functions (Cassidy 1994).

Environmental factors appear to play a significant role in birth defects. A study of 9,000 women whose first child had a birth defect found that when the woman moved to a new city, she was only half as likely to have a second child with a birth defect (Lie 1994). The authors concluded that important teratogens in the environment have yet to be identified.

Some researchers believe estrogenic pollutants, which include some pesticides, may already be causing adverse effects in infants (Sharpe and Skakkebaek 1993, Sharpe 1995). A doubling of the incidence of undescended testes, or cryptorchidism, in male infants since 1960 has been reported in the United Kingdom (Jackson 1988) and increased incidence of urethral abnormalities, known as hypospadias, has also been reported in male infants in recent decades (Giwercman 1992).

A new report suggests that increases in male reproductive disorders in humans and wildlife may be due to exposure to DDT (Kelce 1995). The researchers determined that DDE, the metabolite of DDT, can block the normal binding of testosterone to its receptor. In studies of laboratory rats, they found a link between DDE exposure and abnormal sexual development in male offspring, such as delayed puberty, nipples on the chest, and smaller seminal vesicles and prostates.

IMMUNE EFFECTS. Scant information is available on the immunotoxicity of pesticides. However, we know it is biologically plausible and there's some evidence to suggest that exposure to pesticides could cause immune dysfunction. In a study of 50 women, those who regularly consumed low levels of aldicarb in their drinking water were found to have increased numbers of T8 cells and elevated stimulation responses to an antigen, when compared with unexposed women (Fiore 1986). In animal studies, pesti-

cides have been linked to a variety of immune defects, including decreased host resistance to infections, suppressed T-cell activity, enhanced B- and T-cell immune response, and contact hypersensitivity (Thomas 1990).

SPECIAL SENSITIVITY OF THE YOUNG. There's an appalling lack of data on the specific toxicity of pesticides on children and on juvenile animals. We do know, however, that the young differ in their ability to metabolize, detoxify, and excrete toxic chemicals. Thus, they are likely to be more sensitive than adults to some pesticides. Studies in rats suggest that metabolic immaturity makes juveniles more sensitive to the lethal effects of the insecticides parathion (Benke and Murphy 1975) and deltamethrin (Sheets 1994).

There's another difference between juveniles and adults: during infancy and childhood organ systems are still developing. Indeed, there are windows of time when the young are particularly vulnerable to chemical influences — that lesson was painfully learned in the 1960s when European mothers who took the drug thalidomide gave birth to babies with severe limb deformations. During embryonic and fetal development, exposures to certain chemicals can lead to irreversible damage.

It appears that the developing nervous system of children is particularly sensitive to toxic exposures. According to a report by the National Academy of Sciences, "The data strongly suggest that exposure to neurotoxic compounds at levels believed to be safe for adults could result in permanent loss of brain function if it occurred during the prenatal and early childhood period of brain development" (NRC 1993a, p. 61).

For instance, children exhibit neurotoxicity from lead at one half to one third the blood levels that cause neurotoxicity in adults. Three neurotoxic insecticides were found to be more lethal to immature rats than to adult rats (Pope 1991).

Because children are growing and many of their tissues have rapidly dividing cells, they may also be more sensitive to some carcinogens (NRC 1993a, p.70). A study investigating mechanisms of cancer found that young rats were seven times more susceptible than older rats to a potent liver carcinogen (Gray 1991).

It is believed that contact with carcinogens early in life causes neoplastic changes in cells that increase the chances of developing cancer later in life. But even though age at first exposure appears to play a role in chemically induced cancers, the relationship is complex; juvenile animals also appear to be less susceptible than adults to tumors at some sites in the body, such as breast tissue (NRC 1993a, p. 70).

Today, every American comes in contact with a complex stew of chemicals, including pesticides.

Unlike our forbears who lived before the post-World War II era, we are continuously exposed to a mixture of different chemicals in air, water, and food. EPA has identified more than 2,800 pollutants in air and more than 500 synthetic chemical contaminants in drinking water. At least 270 pesticides are directly applied to food crops.

In addition, some chemicals that were banned 20 years ago because of the hazards they pose to health are still with us today. For instance, the U.S. banned the use of the insecticide DDT on crops in 1972, but because it has a half-life of 7 years in the body and tends to accumulate in the food chain, we all carry traces of it in our fatty tissues. DDT also continues to be used in other countries. Likewise, the manufacture of products with PCBs (used as electrical insulators) ceased in the U.S. in 1977, but products made previously are still in use and being discarded. DDT and PCBs are still circulating widely and indeed, they can be found in air, water, soil, plants, animals, and people around the globe.

Since Delaney was adopted, we've learned people face risks from pesticides in more than just food.

The sources of exposure to chemicals in the environment today are many — pesticides occur in food, water, air, on pets, playgrounds, and lawns. Any useful estimate of the American public's exposure must consider all of these sources.

Furthermore, exposure estimates are only

accurate for pesticides with the same mechanism of action and target organ. Scientists now know that these compounds generally combine to cause an effect. For instance, a recent study found that a mixture of 10 estrogenic compounds, each at one-tenth the dose needed to individually cause an effect, acted in concert to cause an estrogenic effect (Soto 1994). Therefore, one might conclude that these 10 compunds are acting on the same target organ with the same mechanism of action.

For unrelated compounds, however, we still know little about how they influence one another's toxicity in the body. We do know that pairs of chemicals sometimes act synergistically to enhance their individual effects. That's why there are warnings of drug interactions on the labels of medicines. Compounds in the environment can also interact in this way. Asbestos and cigarette smoke, for instance, enhance each other's cancer-causing potential in the body. The insecticides malathion and EPN are also more toxic taken together than they are alone, although this information has not been used in setting "safe" residue levels in foods (NRC 1993a, p. 342).

Unfortunately, there are significant gaps in the toxicity data available for chemicals, making it difficult to know which compounds should be considered together in estimating exposure. And no one can say how many different chemicals the "average" American actually comes in contact with each day.

While researchers now have a much better idea how to accurately assess human exposure to chemicals in the environment, on a practical level, it remains very difficult to do. In essence, we've learned that there are serious flaws in the way exposure estimates were made in the past and are still made today.

Since Delaney was adopted, we've learned that infants and children receive higher exposures to pesticides than do most adults.

When compared with adults, infants and children are likely to receive higher exposures to pesticides because of their small body size and because of how they interact with the environment around them. For instance, young children are more likely to come in contact with pesticides sprayed in the home because chemicals linger longer at ground level; children also pick up pesticide residues while playing on chemically treated lawns and with pets treated for fleas and ticks (Needleman 1994, pp. 120-125).

The peculiar eating habits of infants and children also increase their chances of ingesting pesticide residues in foods. According to USDA national food consumption surveys, children of ages one through five eat about three times more food per unit of body weight than the average American. They also consume relatively large volumes of water, often in infant formulas and in reconstituted juices.

The typical diet of infants and young children is also less diverse than that of adults and includes a high proportion of fruits. USDA has estimated that the average one-year-old drinks 21 times more apple juice, 11 times more grape juice, and about 5 times more orange juice per unit of body weight than the average American. A one-year-old's consumption of grapes, bananas, peanuts, apples, pears, broccoli, strawberries, carrots, tomatoes, potatoes, and peaches is from 2 to 7.5 times greater than the national mean.

These data suggest that infants and young children may be ingesting larger quantities of pesticide residues from fruits and vegetables than previously realized. Data from the EPA shows that infants and young children are permitted to have dietary exposures to potentially carcinogenic and neurotoxic pesticides in amounts that exceed published standards by a factor of 1,000 (Fisher 1992).

Indeed, a committee of the National Academy of Sciences calculated that some children with average diets currently receive a dose of neurotoxic pesticides in their food that exceeds the EPA's "safe" dose. The committee estimated the exposure of 2-year-old children to five commonly used organophosphate insecticides — acephate, chlopyrifos, dimethoate, disulfoton, and ethion.

Using data on the level of residues found on eight foods (apples, oranges, grapes, beans, tomatoes, lettuce, peaches, and peas) and three juices (apple, orange, and grape) commonly consumed by toddlers, the committee determined that 1.3 percent — or about 50,000 American 2-year-olds — receive a combined dose of these five insecticides that exceeds EPA's "safe" dose for organophosphates (NRC 1993a, pp. 305-307). This estimate does not take into account all sources of exposure to all organophosphate pesticides, nor does it factor in the increased sensitivity of young children to neurotoxicants.

Furthermore, exposure to carcinogenic pesticides in raw fruits and vegetables occurs disproportionately during infancy and childhood. For instance, EWG calculated that by age 6 the average American child has accumulated about 35 percent of his or her allowable lifetime cancer risk from captan, a fungicide frequently used on apples, grapes, and peaches and considered to be a probable human carcinogen (EWG 1993, pp. 39-41).

In setting food safety standards for carcinogens, EPA calculates the dose that should raise an individual's risk of cancer by no more than one in a million over a lifetime. However, the agency incorrectly assumes that exposure occurs evenly over the years and that by age 6 a child will have received only 8 percent of the lifetime cancer risk from captan.

The Delaney Clause does not go far enough.

The Delaney Clause is an amendment to the Federal Food Drug and Cosmetic (FFDC) Act of 1954. The original FFDC Act, in section 408, authorizes the setting of "tolerances," or allowable concentrations of pesticide residues in or on raw agricultural commodities. These tolerances are to be set at levels that protect public health, but regulators may also consider the need for "an adequate, wholesome and economical food supply." In other words, when setting standards for pesticide residues in raw foods, the law allows public health risks to be weighed against the public access to nutritious and aforadble food.

The Delaney amendment to section 409, which governs food additives, specifically applies to pesticide residues in processed foods. The clause prohibits the addition of any pesticide that induces cancer in humans or animals if the pesticide becomes more concentrated during processing.

Thus, there's an inconsistency: for processed foods, there's a zero-risk standard for a carcinogenic pesticide; for raw fruits and vegetables, however, the same carcinogenic pesticide is allowed if the economic benefits of using the pesticide are judged to outweigh the risks to health. Another nonsensical result of this legislation is that a weakly carcinogenic pesticide may be excluded from processed foods, while a very potent neurotoxic pesticide would be allowed.

Interpreting these complex and contradictory rules is not easy, and EPA has had to divert significant resources toward deciding when to apply which rule (NRC 1987, p.22). For instance, in order to implement the law, the agency must often determine whether a pesticide residue will concentrate in a food product during processing.

In practice, EPA has often chosen not to enforce the Delaney Clause at all, in part because it is difficult to know whether a vegetable will wind up in a can or in the produce section of the supermarket. Instead, the agency sets tolerances for carcinogenic pesticides based on a "negligible risk" standard — that is, a dose that should raise an individual's lifetime risk of cancer by no more than one in a million. This practice by the agency was challenged by Natural Resources Defense Council and in February 1995, a settlement was reached requiring that EPA revoke some uses of 37 carcinogenic pesticides.

At present, EPA allows residues in food of approximately 70 pesticides that are carcinogenic in animals, and of approximately 20 pesticides that are considered probable human carcinogens.

Although the goal of the Delaney Clause remains critical to public health policy and should remain as the cornerstone of pesticide

policy, our improved understanding of carcinogenesis and of chemical toxicity now enables us to be more efficient in applying the principle of prevention it embodies.

When the Delaney Clause was adopted, there was still much to learn about how certain chemicals triggered cancer. Like most things in science, models to explain carcinogenesis started out simple and have become more complex. Researchers now know that there are a number of different mechanisms involved in the development of chemically induced cancers. And they know that chemicals can play a variety of roles in the processes leading up to cancer. For instance, some chemicals called mutagens cause direct genetic damage that can lead to mutations and eventually to runaway cell division. Other chemicals appear to indirectly enhance the potential for cancer by altering hormone levels or by suppressing immunity.

Not all cancer-causing chemicals are equivalent in their potential to do harm. Today scientists can rank carcinogens by potency. But the Delaney Clause does not allow regulators to distinguish carcinogens by potency nor to focus on those chemicals that are highly carcinogenic and most hazardous.

Furthermore, the law focuses too narrowly on cancer as a potential health effect of dietary exposure to pesticides. Since 1958, the field of toxicology has moved beyond cancer. Researchers now have tests to evaluate the effects of chemicals on fetal development and on the nervous, immune, and reproductive systems. We now know that pesticides can have adverse effects on any or all of these systems. But the Delaney Clause does not reflect the modern view that protecting the public from exposure to toxic chemicals means more than just protecting them from carcinogens. Thus, the Delaney Clause does not allow regulators to use the best science available.

The Delaney Clause and related food safety regulations do not adequately protect public health.

While the Delaney Clause is often called a strict law, on closer inspection it fails to meet its own goal of protecting the public from dietary carcinogens because the law does not apply to most of the food supply. The public eats a wide variety of agricultural products, but the Delaney Clause only addresses processed foods in which pesticide residues concentrate.

According to a report by the National Research Council, "At most, the Delaney Clause could apply to processed-food residues responsible for only one-fifth of the estimated dietary oncogenic risk from pesticides" (NRC 1987, p. 5). The NRC report concluded that foods accounting for nearly half of the total dietary oncogenic risk (all meat, milk, poultry, and pork products and many fruits and vegetables) are beyond the scope of regulation under the Delaney Clause.

Most pesticide residues in foods are not regulated by the Delaney Clause; instead, the EPA sets acceptable tolerances for them in accordance with section 408 of the FFDC Act. Although the law states that tolerances are to balance public health risks against the economic benefits of pesticide use, in practice it appears that public health is not given enough weight. According to a committee of the National Academy of Sciences (NRC 1993a, p. 2):

> "Tolerance concentrations are based primarily on the results of field trials conducted by pesticide manufacturers and are designed to reflect the highest residue concentrations likely under normal conditions of agricultural use. Their principle purpose is to ensure compliance with good agricultural practice. Tolerances are not based primarily on health considerations."

There are a number of other shortcomings in the tolerance-setting process — due largely to incorrect assumptions about exposure — that further weakens the likelihood that public health will be protected. For instance, when EPA evaluates the health risks of a pesticide, the agency assumes that only one residue occurs at a time on a serving of food. In reality, multiple pesticide residues commonly occur in single servings.

One can find single samples of peaches, celery, and carrots with three carcinogens; single samples of apples with three neurotoxicants; and single samples of potatoes with two endocrine-disrupting pesticides (EWG 1994a). While one pesticide residue may pose only a negligible risk to consumers, the so-called safe dose may be exceeded when many pesticides with similar toxic effects are added up.

Likewise, EPA does not consider that some members of the public are also exposed to pesticides in drinking water and in the garden and home, which could all contribute to an unsafe dose. Children, in particular, may receive heavier pesticide exposures, but current practice is to assess health risks based on exposure estimates for the "average adult." Current law does not require that pesticide residues in foods are safe for children, the most vulnerable members of society.

The problem with current law is that it reflects tunnel vision — the big picture is ignored and chemicals are regulated as if they occurred separately. In reality, tens of thousands of different toxic chemicals — trillions of pounds of them — are dispersed into the environment each year. In most cases, these releases are perfectly legal because our laws allow an unlimited number of toxic chemicals to be dumped into the environment, so long as each individual compound does not exceed the "safe" dose.

Trouble is, even the most rigorous scientific analysis cannot predict a "safe" dose of a toxic compound when it occurs in combination with so many others. Little is known about the human health effects of chronic exposure to such an array of chemicals, although there's evidence that related compounds often act additively and some chemicals even enhance one another's toxicity.

In practical terms, our laws are producing ready-to-eat apples with eight different pesticides on them, air in most cities with hundreds of pollutants in it, and treated tap water in parts of the Midwest with up to five different cancer-causing herbicides. Common sense tells us that we cannot continue to dump tons of toxic chemicals into the environment, continue to regulate their releases individually, and expect to escape harming human health.

A prevention approach to regulating pesticides in food would be cost-effective and protect public health over the long-term.

A basic tenet of public health is to prevent disease at the source, rather than managing or treating it after it occurs. This approach makes sense in the long-run. As childhood immunization programs have shown, preventing disease, as opposed to treating sick people, saves health care dollars and is more humane.

A prevention philosophy can also be effectively used to tackle the problem of pollution and its impact on health. By reducing exposure to toxic chemicals, we can prevent illness. For instance, the banning of lead in gasoline, interior house paint, and food cans has lowered blood lead levels in children around the country. Few would dispute the wisdom of preventing exposure to lead, considering the alternative: dealing with neurological impairment in potentially millions of children and, ultimately, adults.

In the spirit of prevention, we should now be phasing out the use of highly toxic pesticides. It's an achievable goal. A relatively small number of carcinogenic, neurotoxic, and hormonally active pesticides contribute to a large fraction of the total hazard that pesticides pose to the public. According to a committee of the National Academy of Sciences, "dietary oncogenic risk appears to be concentrated in a relatively small number of pesticides and crops" (NRC 1987, p.6). The National Academy of Sciences committee made a risk estimate of 28 compounds in 178 food uses and found that 80 percent of the estimated dietary oncogenic risk came from residues of 10 pesticides on only 15 different foods. The group concluded, "Logic argues that the EPA should focus its energies on reducing risk from the most worrisome pesticides on the most consumed crops, and compelling reasons support such a strategy" (NRC 1987, p.14).

There's no need to accept the health risks

associated with uses of the most highly toxic compounds. We can replace them with better solutions to the pest problem; there are plenty of available alternative pesticides and pest control technologies for farmers of every crop in the U.S. (NRC 1989). By phasing out the most hazardous pesticides, we will also encourage the development of new technologies and create markets for safer approaches.

A policy of prevention will not only strengthen protection of public health, it will also curb the spread of pollution. That should save taxpayer money now spent on expensive, and often unsuccessful, efforts to clean up degraded air, soil, and water.

Indeed, while most of the benefits of pesticide use go to producers (and chemical companies), most of the costs are passed on to the public (Pimentel 1992). An analysis of the total cost of pesticide use found that producers' annual investment of $4 billion on pesticides for agriculture provides them with a $16 billion profit. What is often overlooked, however, is that the environmental and social costs are about $8 billion (Pimentel 1992). Most of that $8 billion cost — in public health impacts; groundwater contamination; losses of domestic animals; costs of enforcing government regulations; losses of fish, birds, and honeybees; losses of natural pest-controlling species and the development of resistant pests — is borne by society at large.

The public should not be required to subsidize the use of massive quantities of highly toxic pesticides on crops and then pay again to remedy the environmental damage they cause. Moreover, as a matter of policy, it is inappropriate that exposed individuals be saddled with the burden of proving that their health has been harmed, especially when it is nearly impossible to show a specific cause-and-effect link between any particular environmental exposure and an individual's health problem.

As part of a prevention strategy, we must demand that chemical manufacturers provide more complete toxicity tests before a product is released into the environment. That's the first line of defense in keeping dangerous compounds out of food, air, and water.

Policy Recommendations

We're ready for a new approach to pesticide regulation that blends common sense with good science to reduce the growing load of toxic chemicals in the environment. A strategy of prevention will take us far beyond Delaney both in protecting public health and in minimizing costs.

We may never acquire complete toxicity information on all the chemicals now released into the environment and we will be hard-pressed to discover how exposure to such a complex chemical stew impacts health. At present only about 20 percent of pesticides registered in the U.S. have complete health and safety information on file at the federal level (Trichilo and Schmidt 1991). Data are also sorely lacking on the specific toxicity of pesticides on infants and juveniles (NRC 1993a, p.4).

Given these uncertainties, it makes more sense to direct our efforts toward preventing exposure to pesticides that are known to be highly toxic in small quantities. We need to set priorities. In fact, the model now commonly used by regulatory agencies to assess a carcinogen's risk to health does not provide a true estimate of risk to health; rather, the model "allows a crude rank ordering of animal carcinogens from the most potent to the least potent, which might then provide a basis for priorities in regulation and pollution prevention" (NRC 1993b, p. 50).

Let's use our scientific knowledge more appropriately to establish which chemicals are simply too hazardous for widespread use. If a substance is highly toxic, let's phase it out and find an alternative, instead of carrying out endless risk assessments based on dubious estimates of the public's exposure.

The idea of phasing out a substance primarily because of its chemical characteristics and potential for doing harm is not new. One important reason for banning DDT was that it accumulates in the fatty tissues of organisms and thus, concentrates in the food chain. Indeed, in 1994 the

International Joint Commission on the Great Lakes (IJC), a U.S. and Canadian treaty organization responsible for monitoring the Great Lakes environment, made strict recommendations to further protect water, wildlife, and people. Aiming for zero discharge of all persistent toxic substances, the IJC called for banning their manufacture, distribution, storage, use and disposal.

We need to follow the IJC's lead and create a new pesticide policy that will:

- ❖ Focus on a pesticide's toxicity, not on a calculation of risk to health;
- ❖ Evaluate a pesticide's toxicity for its potential effects on the most sensitive members of the population, i.e., embryos, fetuses, infants and children;
- ❖ Evaluate all potential toxic effects of a pesticide, including its ability to cause cancer, its neurotoxicity, immunotoxicity, and effects on reproduction and development;
- ❖ Target the most potent and toxic pesticides for phase-out within a reasonably short period of time.

Because pesticides provide many benefits to society, the criteria for phase-outs must be prudent. A chemical should be phased out that meets one or more of the following criteria:

- ❖ The disease or effect we are protecting against is most often delayed, irreversible, or severely debilitating (e.g., cancer, chronic nervous system damage, birth defects);
- ❖ The weight of the evidence strongly indicates that the disease or effect occurs consistently in animal studies, with a strong dose-response relationship in exposed animal populations (e.g., B2 carcinogens and most strong reproductive toxicants);
- ❖ There is no threshold of safe exposure yet identified and employed by federal agencies (e.g., B2 carcinogens, many endocrine disruptors);
- ❖ The compound persists in the environment or concentrates in the food chain.

The burden of conducting complete toxicity tests should be placed squarely on the shoulders of pesticide manufacturers, not on public agencies. As for pesticides that are judged safe enough to allow their continued use on food crops, EPA should set strict food safety standards that explicitly protect the health of infants and young children. Furthermore, farmers should be rewarded for reducing their use of pesticides. All of these measures will enhance the effectiveness of a new policy of prevention that moves beyond Delaney.

An ounce of prevention is worth a ton of risk management.

Alternatives To High Hazard Pesticides

Over the past twenty years, American taxpayers have spent hundreds of millions of dollars on research identifying biological pest controls, developing methods to gauge economically threatening levels of pest populations, locating vulnerable ecological areas, and calculating resistance in pest species. Precious little effort, however, has been directed at integrating this disjointed research into workable pest control systems, and even less energy has been devoted to reversing economic and regulatory incentives that reward current inefficient levels of pesticide use.

In 1979, the Office of Technology Assessment estimated that U.S. farmers could reduce pesticide use by 75 percent simply by adopting integrated pest control measures available at that time. The report was virtually ignored by American farmers. Ten years later, the National Academy of Sciences completed an exhaustive review of farming systems in the United States, concluding that, "Farmers who adopt alternative farming practices often have productive and profitable operations, even though these farms usually function with little help from commodity income and price support programs or extension" (NRC 1989).

According to the Academy:

> "Well-managed alternative farming systems nearly always use less synthetic chemical pesticides, fertilizers, and antibiotics per unit of production than comparable conventional farms. Reduced use of these inputs lowers production costs and lessens agriculture's potential for adverse environmental and health effects without necessarily decreasing — and in some cases increasing — per acre crop yields and the productivity of crop livestock systems" (NRC 1989).

It is abundantly clear that American agriculture would survive and even flourish under a public health driven pesticide policy that phased-out highly hazardous pesticides and implemented a strict standard of protection for children. In fact, thousands of commercial scale growers of staple food commodities have done that and even more, with little assistance from the government. Some of the best examples include:

Practical Farmers of Iowa has helped hundreds of mainstream commercial corn, soybean, and hog producers substantially reduce and in some cases eliminate the use of synthetic chemical pesticides and fertilizers. These gains are typically achieved through rotations, comprehensive monitoring of nutrient availability from all sources, diversification, and a commitment to on-farm research. Most of these farmers increase their income by reducing input costs, maintaining or even increasing yields, and developing specialty crops as new sources of income.

The BIOS project, an integrated effort to promote biologically based integrated pest management that is less reliant on chemical pesticides, eliminated insecticide use and cut herbicide use in half in one year on thirty commercial almond orchards in Merced county, California. Based on this success, BIOS, which involves growers, university researchers, seed and input suppliers, advocates of sustainable agriculture, the state of California, and the almond industry, is expanding to other counties and at least five additional crops this year.

Green Cay Farms in Florida has cut pest control costs in green peppers by 63 percent and eliminated all but one chemical pesticide application through the introduction of a biologically based pest control program that integrates predator insects to control thrips, plant-

ing of cover crops for predator habitat, and the use of biological pesticide sprays.

Organic cotton growers in California alone raised 11,000 acres of organic (or transition to organic) cotton in 1993. Through modification in planting techniques (less plant density per acre), composting, applications of foliar nutrients, and the release of biological pest controls, organic growers produce the same yields per acre with less than half the costs for pest control as cotton grown with synthetic chemical pesticides and fertilizers.

Tom and Steve Pavich currently grow several thousand acres of organic grapes, making them one of the largest grape producers in the country. The Pavich operation has become extremely profitable by producing consistently higher yields per acre than conventional growers, with marginally higher production costs, after eliminating all synthetic chemical pesticides and fertilizer use in their vineyards.

Even though hundreds of millions of taxpayer dollars have been spent in the past twenty years on research directly relevant to pesticide use reduction, this is but a minuscule portion of overall research dollars at the U.S, Department of Agriculture (USDA). Out of a $1.4 billion annual USDA research budget, no more than $15 million is devoted to research and promotion of farming systems that lower pesticide use and move farmers toward more biologically integrated systems of farming.

There are many more examples of success in reducing and eliminating synthetic chemical pesticide use in U.S. agriculture. Nonetheless, these growers remain in the minority. An essential component of any policy to encourage agricultural innovation and adoption of systems like these is public health-based pesticide policy that eliminates the use of high hazard pesticides over a reasonable period of time, complimented by a tough health standard that protects children, from those pesticides that remain on the market.

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